ETS – Environmental Tobacco Smoke

Report from a workshop on effects and exposure levels March 15-17, 1983, Geneva, Switzerland

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Preface

The Second Workshop on Environmental Tobacco Smoke with particular reference to effects and exposure levels was held in Geneva, Switzerland, March 15-17, 1983.

The workshop was organized by Ragnar Rylander M. D, University of Gothenburg, Sweden, and University of Geneva, Switzerland, together with Yvonne Peterson and Marie-Claire Snella, research assistants and Isabelle Gourdon. It was supported by a grant from the Tobacco Institute, Washington D. C., to the University of Geneva. The symbol for the workshop was designed by Ariane Catry.

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3.6. The effect of environmental tobacco smoke in two urban communities in the west of Scotland

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INTRODUCTION

The question of whether environmental tobacco smoke (ETS) can damage health has not yet been clearly answered. It is known that a lighted cigarette emits more sidestream smoke than mainstream and that the smoke available for involuntary inhalation contains substantial amounts of carbon monoxide, tar, nicotine, benzo(a)pyrene and other carcinogens, and oxides of nitrogen (1).

Studies from Japan (2) and Greece (3) have suggested that non-smoking wives of heavy smokers have a two-fold increased risk of lung cancer when compared with non-smoking wives of non-smokers. In contrast, analysis of data from the prospective study of the American Cancer Society volunteers (4) has suggested that very little, if any, increased risk of lung cancer exists when non-smoking women married to smoking husbands and non-smokers married to non-smoking husbands are compared.

The present study has been carried out in a defined population group in an area of high incidence (5) of lung cancer with a precisely defined population base. It reports lung cancer data on both males and females.

MATERIALS AND METHODS

The study comprises 16,171 apparently healthy individuals aged between 45 and 64, resident in Renfrew and Paisley, two urban areas in the West of Scotland. They took part in a multiphasic screening survey for cardiorespiratory disease between 1972 and 1976. This represented a response rate of 80 % of those randomly sampled from the resident population. Details of this survey have been described by VMH (6). Information on each respondent's smoking habits and their experience of symptoms of respiratory and cardiovascular disease were collected using a self-completed questionnaire, carefully checked at the time of attendance at the screening unit.

The diagnosis of cancer in each individual has been checked in the West of Scotland Cancer Registry and follow up for mortality carried out by record linkage (7) with data from the Registrar General for Scotland. Follow up is complete until 31 December 1982.

As members of the same household attended the screening unit, it was possible to identify smoking and non-smoking partners of smokers and non-smokers. These were allocated to categories defined so as to represent an increasing measure of tobacco exposure.

TABLE 1. *Number and percentage of individuals by category.*
Number of individuals attending screening = 16,171
Number within partnerships screened = 8,128 (ex-smokers excluded)

Category	Male N	Male %	Female N	Female %
Controls	517	12.7	523	12.9
ETS exposure	310	7.6	1394	34.3
Smoking	1395	34.3	310	7.6
Smoking + ETS exposure	1845	45.4	1834	45.2
Total	4067	100	4061	100

TABLE 2. *Age standardized prevalence of self-reported respiratory symptoms by category. Per cent of all within each group. Males*

Respiratory symptom	Controls	ETS exposure	Smoking	Smoking + ETS exposure
Infected spit	3.3	4.2	11.1	12.5
Persistent spit	10.1	14.5*	33.9	35.6
Dyspnoea	7.4	11.9*	14.0	15.4
Hypersecretion	7.2	11.9*	20.6	21.6
Number of individuals	517	310	1395	1845

*P value < 0.05 for comparison of control and ETS exposure group

TABLE 3. *Age standardized prevalence of self-reported respiratory symptoms by category. Per cent of all within each group. Females*

Respiratory symptom	Controls	ETS exposure	Smoking	Smoking + ETS exposure
Infected spit	2.1	2.8	10.0	9.1
Persistent spit	6.3	7.2	23.9	23.1
Dyspnoea	9.7	14.7**	16.2	18.3
Hypersecretion	3.9	4.8	17.6	17.1
Number of individuals	523	1394	310	1834

**P value < 0.01 for comparison of control and ETS exposure group

1. Control—an individual who does not smoke and who lives at the same address as another individual who does not smoke.
2. ETS exposed—an individual who does not smoke but who lives at the same address as another individual who does smoke.
3. Smoker—an individual who is a smoker or who has given up smoking up to five years ago but who lives at the same address as an individual who does not smoke.
4. Smoker and ETS exposed—an individual who is or who has been a smoker up to five years ago and who lives at the same address as an individual who also smokes.

All individuals in these categories were aged 45-64 at the time of the survey. Ex-smokers who had given up smoking for five years or more have been excluded from this analysis.

RESULTS

The number of males and females in each of the categories defined above is shown in Table 1. 97.6% of the pairings were male/female partnerships.

The prevalence of self-reported respiratory symptoms (6) found at the survey is shown for each category for males in Table 2 and for females in Table 3. For each measure, infected spit, persistent spit, dyspnoea and hypersecretion an increasing dose response relationship was evident in males. The prevalence of these four symptoms was slightly higher in the exposed to ETS than in the controls. This observation was consistent in both males and females.

The prevalence of cardiovascular symptoms found at the time of the survey is shown in Table 4. In females angina and ECG abnormalities (6) were slightly more common in the group exposed to ETS than in the controls, although the magnitude of the differences was small. The reverse trend was shown for males.

Male mortality for the different categories is shown in Table 5. A dose-response relationship was found for lung cancer rising from a rate of 4 per 10,000 for the control group to 13 per 10,000 for the group exposed to ETS to 22 per 10,000 for the smoking group and 24 per 10,000 for the smoking group also exposed to ETS. The rates for other smoking related cancers and for smoking related diseases (8) did not show a difference between the control and groups exposed to ETS except for the rate for myocardial infarction (ICD410) which was

TABLE 4. *Age standardised prevalence of cardiovascular symptoms by category. Per cent of all within each group*

Cardiovascular symptom	Controls	ETS exposure	Smoking	Smoking + ETS exposure
Males:				
Angina	6.6	6.4	9.6	12.3
Major ECG abnormality	1.4	1.3	2.0	2.2
Females:				
Angina	4.2	5.3	5.4	6.1
Major ECG abnormality	0.4	0.6	0.6	0.5

TABLE 5. *Annual age standardised mortality rates per 10,000 by smoking category Males*

Cause of death	Controls	ETS exposure	Smoking	Smoking + ETS exposure
All causes	91	90	156	156
Lung ca	4(2)	13(4)	22(30)	25(44)
Other Ca	12(6)	6(2)	24(34)	22(41)
MI (410)	31(16)	45(14)	60(84)	46(84)
IHD (411-4)	4(2)	0(0)	11(15)	14(25)
CVD	10(5)	3(1)	12(17)	16(29)
Others	31(16)	23(7)	27(38)	35(64)
Smoking related	75(39)	77(24)	140(195)	134(247)
Non-smoking related	16(8)	13(4)	17(23)	22(40)
Total number of deaths	47	28	218	287

Figures in parenthesis are the numbers of deaths

TABLE 6. *Annual age standardised mortality rates per 10,000 by smoking category Females*

Cause of deaths	Controls	ETS exposure	Smoking	Smoking + ETS exposure
All causes	40	58	87	77
Lung Ca	4(2)	4(6)	7(2)	6(11)
Other Ca	19(10)	24(33)	26(8)	22(40)
MI (410)	4(2)	12(17)	19(6)	21(39)
IHD (411-4)	0(0)	1(2)	3(1)	2(4)
CVD	2(1)	4(5)	7(2)	9(16)
Others	12(6)	13(18)	26(8)	17(31)
Smoking related	15(8)	30(42)	55(17)	52(96)
Non-smoking related	23(12)	27(37)	36(11)	24(44)
Total number of deaths	21	81	27	141

Figures in parenthesis are the numbers of deaths

TABLE 7. *Percentage smoking 15 or more cigarettes per day*

	Controls	ETS exposure	Smoking	Smoking + ETS exposure
Males	0	0	41.8	57.3
Females	0	0	46.5	53.4

slightly higher in the group exposed to ETS than in the controls.

Female mortality is shown in Table 6. All causes mortality is higher in the group exposed to ETS than in the controls. This was not the case for lung cancer although mortality from myocardial infarction was higher in the group exposed to ETS when compared with the controls.

Division of all diseases into those considered smoking and non-smoking related (8) produced a higher rate in the group exposed to ETS when compared with controls.

On account of the apparently unusual relationship between lung cancer risk and tobacco consumption in the West of Scotland (9) the amount smoked by individuals in the defined categories is shown in Table 7. In the smoking group also exposed to ETS 57.3 % of males and 53.4 % of females smoked more than 15 cigarettes per day. This compares with 41.8 % of males and 46.5 % of females in the smoking group.

DISCUSSION

Insufficient time has elapsed since the completion of the recruitment phase of this study (1976) for sufficient numbers, either of incident cases of cancer or of other diseases, to allow firm conclusions to be based on the results. The results have been expressed as annual age standardised rates per 10,000, as the total number of incident cases and the number of deaths is small in the control and ETS exposure groups (Tables 5, 6).

The results relate to only 8,128 of the 16,171 individuals who attended the multi-phasic screening unit (50 %). Some of this discrepancy can be accounted for by those living alone, those living with a partner outwith the age range, and those living with a partner who has not attended. Those who have been exsmokers for five years or more were also excluded from the analysis. As there is still doubt whether these groups account for the total discrepancy, given an initial response rate of 80 %, the authors require to continue their investigation of this apparent discrepancy.

This study has unique features which allow even preliminary results to be of interest.

These are:

1. The study has been carried out in an area with the highest national incidence rate of lung cancer recorded (5).
2. It is a prospective cohort study carried out in a geographically defined population whose members are homogeneous by social class and ethnic group.
3. Other reports (2, 3, 4) concentrate on females. This study includes both sexes.
4. No questions concerning exposure to ETS were asked, thus avoiding the bias inherent in self-reported assessments of partnership dosage.

Given the strength of the epidemiological association between cigarette smoking and lung cancer, it is this disease rather than ischaemic heart disease that would be first to appear in excess in the cohort if a dose response relationship existed, especially as the respondents were all apparently healthy at the time of screening.

In males, the cases of lung cancer occurring in non-smokers were found more frequently in those exposed to ETS (4/310) than in the controls (2/517) (Table 5). No dose-response relationship was apparent in females for lung cancer deaths though an effect was present when all smoking related (8) deaths including deaths from myocardial infarction were taken into account (Table 6).

These findings may be supported to an extent by the dose-response relationship that exists for self-reported respiratory symptoms

(Tables 2, 3), all of which are more frequently reported in the group exposed to ETS than in the controls and four of which achieve statistical significance.

The number of deaths in the control and ETS exposure groups is very small and may explain the lack of an apparent dose-response in females. However, as the relative risk for lung cancer for active smokers is much higher in males than females it may be too early to expect many females in the ETS exposure group to be affected. This would also apply to male as well as female deaths from myocardial infarction.

Occupation has not been taken into account in this analysis, as its effect on lung cancer risk in non-smokers is thought to be marginal (4, 10).

The West of Scotland is a valuable area to continue examination of the effect of ETS on account of the relatively high rate of lung cancer in non-smokers and the flattening of the dose-response relationship above an average consumption of 20 cigarettes per day (9).

In conclusion, the clear dose-response relationship with lung cancer observed in males exposed to ETS supports observations from previous studies. Although the number of deaths on which the current analysis is based is small. The nature of the findings makes continuation of this study important.

REFERENCES

1. US Department of Health, Education and Welfare. Smoking and Health: A report of the Surgeon General, Washington, DC: US Public Health Service 1979.
2. Hirayama T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. Br Med J 1981: 282:183-185.
3. Trichopoulos D, Kalandidi A, Sparros L, McMahon B. Lung Cancer and Passive Smoking. Int J Cancer 1981: 27:1-4.
4. Garfinkel L. Time trends in lung cancer mortality among non-smokers and a note on passive smoking. J natn Cancer Inst 1981: 66:1061-1066.
5. Gillis C. R, Boyle P, Hole D. J, Graham A. Cancer incidence in UK, Scotland West 1975-1977. *In* Waterhouse J, Muir C, Shanmugaratnam K, Powell J (Eds), Cancer incidence in five continents, Volume IV, Lyon, IARC Scientific Publications No 42, 1982.
6. Hawthorne V. M, Greaves D. A, Beevers D. G. Blood pressure in a Scottish town. Br Med J 1974: 269:600-603.
7. Hole D. J, Clarke J. A, Hawthorne V. M, Murdoch R. M. Cohort follow-up using computer linkage with routinely collected data. J chron Dis 1981: 34:291-297.
8. Doll R, Peto R. Mortality in relation to smoking: 20 years observations in male British doctors. Br Med J 1976: 273:1525-1536.
9. Gillis C. R, Hole D. J, Hawthorne V. M, Boyle P. Male lung cancer and cigarette smoking in the West of Scotland (submitted).
10. Friedman G. D, Petitti D. B, Bawol R. D. Prevalence and correlates of passive smoking. Am J publ Hlth 1983: 73:401-405.
11. Office of Census and Surveys. Occupational mortality: The Registrar General's decennial supplement for England and Wales. 1970-72. London: HMSD 1978.

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